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DOES ONE-YEAR OUTCOME AFTER ACUTE MYOCARDIAL INFARCTION DIFFER WITH RACE? RESULTS FROM THE TIMI II TRIAL

Bernard R. Chaitman, Morton J. Kern, Herman A. Taylor, Frank V. Aguirre, George Sopko, Richard N. Ross, Edwin C. Bovill and TIMI Investigators. St. Louis University School of Medicine, St. Louis, MO

The impact of race on 1-year outcome after acute myocardial infarction (MI) was examined in 2,564 white pts (W), 174 black pts (B), and 147 hispanic pts (H) treated with rt-PA, heparin, and aspirin in TIMI II clinical centers enrolling more than one minority patient.

Results:

	W	B	H	p
N	2,564	174	147	
Sex, male (%)	82.4	71.3	85.7	0.001
Current smoker (%)	49.4	62.1	55.1	0.003
Diabetes (%)	11.9	22.4	19.7	<0.001
Hypertension (%)	36.7	55.8	40.6	<0.001
Fibrinogen Reduction†	109(1092)	151(93)	112(69)	<0.001
EF*	50.6(2091)	48.3(133)	48.9(122)	0.06
3-week mortality (%)	4.8	5.8	3.4	NS
1-year mortality (%)	7.3	12.3	6.3	0.05

†Baseline - 5 hour (mg/dl), mean (n);
*Hospital discharge ejection fraction (%), mean (n)

On protocol angiography, the location and patency of the infarct-related artery were similar in the three groups. One-year mortality after MI treated with thrombolytic therapy may be greater in blacks than whites and hispanics. Blacks have a greater reduction in 5-hour fibrinogen levels after rt-PA therapy than whites or hispanics.

COMPARISON OF IMMEDIATE AND LONG TERM OUTCOME OF PATIENTS HAVING EMERGENT OR DEFERRED CORONARY BYPASS SURGERY FOLLOWING INTRAVENOUS THROMBOLYTIC THERAPY FOR ACUTE MYOCARDIAL INFARCTION

Dean J. Kereiakes, Robert M. Califf, Barry S. George, Joseph Samaha, Sharon Y. Young, Linda C. Anderson, Eric J. Topol, The Christ Hospital, Cincinnati, Ohio

Coronary bypass surgery (CABG) was performed following IV thrombolytic therapy in 303 (22%) of 1387 pts. enrolled in TAMI 1-5 trials. CABG was emergent (E-CABG) in 36 (2.6%) and deferred (D-CABG >24 hrs.) in 267 pts. (19.3%). E-CABG pts. had more frequent preoperative shock (24% vs 6%), and anterior infarction (61% vs 45%) than pts. with D-CABG. Failed PTCA and left main or equivalent coronary disease were more frequent in E-CABG pts. ($p<.0001$). E-CABG was more common in trials using r-tPA monotherapy vs combination r-tPA and urokinase (5.0% vs 0.8%). Comparison of composite adverse endpoints demonstrated more complicated hospital course following E-CABG including death (17% vs 5%), blood transfusion ($p=.001$) and less frequent use of internal mammary grafts ($p=.011$). Significant improvement in left ventricular ejection fraction (LVEF) and infarct zone(IZ) function followed both E or D-CABG. At six month follow-up of hospital survivors, mortality was 4% E-CABG, 3% D-CABG. These findings suggest that failed PTCA is the most frequent indication for E-CABG and is more common following r-tPA monotherapy than combined lytic therapy. E-CABG is associated with more frequent complications than D-CABG including death and transfusion. Significant improvement in LVEF and IZ function follows both E and D-CABG. Thus, CABG should be deferred >24 hrs. following thrombolytic therapy if clinically possible.

INFARCT ARTERY AND LESION MORPHOLOGY IN PATIENTS WITH MINIMAL STENOSIS AFTER THROMBOLYTIC THERAPY FOR ACUTE MI.

GJ Taylor, FL Mikell, DL Koester, A Song, JT Dove, HW Moses, RE Katholi, HA Wellons, JA Schneider. Prairie Education & Research Cooperative, Springfield, IL.

Atherosclerotic plaque, spasm and thrombosis interact in varying degree to produce coronary artery occlusion. Coronary spasm or thrombosis may be more important when stenosis is minimal. In a prospective study of 580 patients having thrombolytic therapy for transmural myocardial infarction (MI) and followed by angiography within 6 days, minimal coronary stenosis (<50% reduction of luminal diameter) was found in 39 pts (7%).

Infarct lesion morphology for those with minimal stenosis fell into 3 categories: (1) No lesion, 8 pts; 2 of these had vasodilator dependence implicating spasm. (2) Ragged, ulcerated plaque, 17 pts, implicating thrombosis. (3) Smooth plaque, 13 pts, implicating neither thrombosis or spasm.

Treatment included antiplatelet and calcium channel blocker therapy. Pts have been followed 51±12 months (range 29-84 months). Reinfarction has occurred in 5 pts (13%), with reinfarction occurring from 1 week to 36 months after MI. Risk of reinfarction was not influenced by lesion morphology.

Conclusion: In pts with minimal stenosis after MI, variable plaque morphology suggests multiple pathogenetic mechanisms for coronary occlusion. While prognosis is good, reinfarction may occur despite treatment with antiplatelet and vasodilator drugs.

PARAMEDIC-INITIATED PRE-HOSPITAL THROMBOLYSIS USING UROKINASE IN ACUTE CORONARY OCCLUSION (T.I.C.O. 2).

Michael O'Rourke, David Gallagher, James Healey, Ken Hillman, Tony McLean, John Hall, Jack Hawkins, Kevin Graham.
St Vincent's, Sutherland, Liverpool, Nepean Hospitals and Central District Ambulance, Sydney, Australia.

Paramedic-initiated thrombolysis carries the best prospect for infarct limitation following thrombotic coronary occlusion. Practicality and safety however have not yet been established. Over a one year period, August '89-'90, we evaluated 1181 patients outside hospital with acute chest pain syndromes. Fifty two fulfilled criteria and 51 consented to receive 2 million units of Urokinase (Ukidan, Serono) as an I.V. bolus. Admission criteria required typical clinical features of acute evolving myocardial infarction within 1.5 (later 3.0) hours of symptom onset, no previous infarction or life threatening illness, age ≤ 72, absence of contraindications to thrombolysis, together with EKG criteria of acute infarction which had yielded 98% specificity and 71% sensitivity in a previous study. Entry also required authorization by cellular phone from a supervising senior cardiologist or intensivist after description of clinical features and the hard copy Marquette PC EKG report with ST segment listing. Time from symptom onset to urokinase injection was 69 (SD 26) minutes. Time saved to thrombolysis was estimated at 60-90 minutes. Diagnosis was confirmed in all but 2 patients (one with right bundle branch block, one without, required reciprocal ST depression). There was one death (L main and R coronary lesions and cardiogenic shock). There were two hypotensive episodes, both associated with bradycardia, and only one minor bleeding complication. APTT (43 SD 17 secs) and thrombin times (32 SD 42 secs) were modestly altered as were indices of fibrinolysis:- FDP 201 (SD 318) µgm/ml, D-Dimer (SD 3.1) µgm/ml, Fibrinogen 1.4 (SD 0.8) gm/L. Coronary artery patency of culprit lesions (TIMI score 2-3) was 66% (19/29) at 15 days (mean). Results indicate that pre-hospital thrombolysis with urokinase is practical and safe.